

**VIA EFS**

**MAIL STOP AMENDMENT**

**IN THE UNITED STATES PATENT AND TRADEMARK OFFICE**

In Re Patent Application of:	:	
Ru Chih C. Huang <i>et al.</i>	:	
	:	
Conf. No.:3871	:	Group Art Unit: 1614
	:	
Appln. No. 10/735,910	:	Examiner: L. Royds
	:	
Filed: December 16, 2003	:	Attorney Docket No.: 2240-199065
	:	
Title: METHOD FOR TREATMENT OF	:	Customer No.
TUMORS USING		
NORDIHYDROGUAIARETIC		
ACID DERIVATIVES		

**DECLARATION OF RAOUL TIBES**

I, Raoul Tibes being over the age of eighteen and having personal knowledge of the facts herein state under oath as follows:

1. I am not an inventor of the present application.
2. Current employment:

TGEN Clinical Research Service

Scottsdale Clinical Research Institute

10510 N. 92<sup>nd</sup> Street, Suite 200

Scottsdale, AZ 85258
3. I have prepared this declaration as a consultant with Erimos Pharmaceuticals, LLC, (the licensee of the patent) through TraCR Consulting, PLLC. TraCR Consulting is a limited liability cooperation. .

4. Academic credentials:

MD, PhD Program Ludwig-Maximilian- University Medical School, Munich

07/00-06/01	Intern in Internal Medicine, New York University Medical Center Residency Program, New York, NY
07/01-06/03	Resident in Internal Medicine, New York University Medical Center Residency Program, New York, NY
07/03-06/06	Fellow in Medical Oncology and Hematology, MD Anderson Cancer Center, Houston, TX
07/04-06/06	Research Fellow, Translational Research in Leukemia, Signal Transduction and Proteomics, Departments of Leukemia and Molecular Therapeutics, U.T. MD Anderson Cancer Center, Houston, TX
01/06-6/06	Fellow in Hematology, MD Anderson Cancer Center and Baylor College of Medicine, Houston, TX, <i>Clinical Rotations in Benign Hematology</i>

My Curriculum Vita is attached.

5. I have 13 years experience in research, six years in cancer research and 3-4 years in drug development and novel therapeutics..

6. This declaration is provided in support of the arguments presented in the Applicants' Response to the Examiner's Final Office Action of July 1, 2009 in which claims 9-10, 15 and 18-20 were rejected as being anticipated under 35 U.S.C. § 102(b).

7. Due to my qualifications and experience within the scientific research field, as well as to how these fields relate to medical conditions, I believe I am a person of skill in the art.

8. I am an investigator on a clinical trial sponsored by Erimos Pharmaceuticals, the licensee of patent application 10/735,910

9. I have reviewed the Examiner's Final Office Action of July 1, 2009 in which claims 9-10, 15 and 18-20 were rejected as being anticipated under 35 U.S.C. § 102(b).

10. **Discussion of Howell et al.**

11. I have reviewed patent 5,541,232 to Howell, et al. ("Howell")

12. Howell does not teach the administration of NDGA and analogs thereof as sole agents to treat leukemia. Howell only teaches the administration of NDGA and analogs thereof together with antineoplastic drugs.

13. Howell is singularly focused on the use of NDGA as an inhibitor of multidrug resistance ("MDR").

14. In my opinion, the data presented by Howell does not support the use of NDGA as a single agent, that is, as an antineoplastic (anti-cancer) agent. Howell lacks data showing that NDGA is effective as a single agent to treat leukemia.

15. Based on the data and assumptions presented by Howell, NDGA should not be given as single agent in to treat leukemia.

16. Howell nowhere in his application assumes single agent activity, nor defines or encompasses a molecular context that would lead a physician to use NDGA as a single agent to treat leukemia. See Howell, col 3 lines 65- col 4, lines 31

17. Howell properly cites studies showing that NDGA alone was ineffective in treating cancer. See Howell, column 4, lines 44-54.

A clinical study was conducted by Smart, et al., reported in U.S. Pat. No. 4,880,637, in which human cancer patients ingested either a tea made from the creosote bush or doses of pure NDGA. This study indicated that neither NDGA nor the tea were effective anticancer agents and in some cases caused stimulation of tumor cell growth. This confirmed the earlier screening studies of NDGA conducted by Leiter, et al. of the Cancer Chemotherapy National Service Center of National Cancer Institute which obtained negative results when NDGA was tested against several types of cancer cells.

18. MDR resistance mechanisms as far as they are known, define a specific molecular and cellular context, that is the presence of various MDR genes/proteins that exert/modify special biological/physiological functions leading to drug resistance to common (cytotoxic)-chemotherapies (or antineoplastic agents/drugs).

19. The field of MDR and clinical testing to overcome such resistance has been extensively studied over the last decades. Howell states that the “present invention contributes to solving the MDR problem”. This again emphasizes that the invention teaches only to overcome MDR resistance. MDR resistance becomes of importance in association with agents used to treat cancer. An MDR modifying agent could have single agent activity. This is however not assumed by Howell to be true for NGDA.

20. In my interpretation, for addressing and overcoming MDR mechanisms in cancer, combination therapy is required. That means a second agent, here the anti-neoplastic agent, need to be present in order for the MDR concept to address what it is trying to overcome: resistance to an anti-neoplastic agent.

21. I am unaware of a single agent regimens that is considered to target MDR as a mechanisms to overcome. I feel that if such an agent would be used alone, it would have single agent activity and not follow the MDR concept, which is to overcome resistance to another antineoplastic agents, as Howell claims to have invented to overcome. This is also reflected in the fact that clinical studies trying to overcome MDR in cancer patients have often used combinations in those studies. This is in contrast to many other novel agents investigated which are studied in the clinic as a single agents,, assuming to find at least some single agent activity.

22. An oncologist reading Howell would understand that Howell clearly rejects the activity of NDGA as single agent and assumes that NDGA does not have antineoplastic activity: “The NDGA compound and antineoplastic agent ..” (column 4, lines 6-9). Further Howell states that “..one or more antineoplastic or cytotoxic agents with NDGA or an analog of NDGA...” (column 4, lines 11-13). Thus Howell itself clearly separates antineoplastic (anti-cancer) activity from the activity of NDGA and does not mention, assume or imply that NDGA does have single agent activity as an antineoplastic agent.

23. Howell’s working examples 1-8 do not contain data suggesting that NDGA works as a single agent to treat leukemia. In examples 2-8 which contain data, the NDGA is administered with a separate antineoplastic agent.

24. Howell does not teach the use of the compounds tetra-O-methyl nordihydroguaiaretic acid or meso-1,4-bis[3,4-(dimethylaminoacetoxy)phenyl]-(2R,3S)dimethylbutane.

25. For the reasons set out above, Howell does not teach the use of NDGA or its derivatives as single agents to treat leukemia.

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26. The mechanism of action against leukemia in the '910 application is not via MDR.

27. The claims of the '910 application are method claims for the treatment of leukemia by administering only the defined NDGA derivatives to a patient in the absence of another antineoplastic agent.

28. As an oncologist in my view, the relevant time period for administration of an active anti-cancer agent to distinguish its activity and therapeutic effect is at least a treatment cycle, the same here applicable for NDGA derivatives.

29. The data presented in the '910 application predict single agent activity.

**Rejection of Claims 9-10, 15 and 18-20 as Anticipated by Howell**

30. I have reviewed the Examiner's Final Office Action of July 1, 2009 in which Claims 9-10, 15 and 18-20 were rejected as being anticipated under 35 U.S.C. § 102(b) over Howell.

31. Claims 9-10, 15 and 18-20 are not anticipated by Howell.

32. I consider Howell to only teach the administration of NDGA in connection with an antineoplastic agent.

33. Due to the lack of data on single agent activity of NDGA, I do not consider Howell to enable the use of NDGA alone as a therapeutic to treat leukemia.

34. The examiner's arguments that NDGA is contemplated as a single agent therapy by Howell because it can be given before or after the antineoplastic drug does in my opinion, not support the use of NDGA as a single agent to treat leukemia. The sequence of administration of

anticancer drugs in combination therapy does not automatically elude or support their single agent activity. Combination therapies in cancer can be given days to up to several weeks apart and still are considered to be part of the same treatment regimen, here defined within one and the same treatment cycle.

35. Treatment of MDR under Howell is by definition the most widely used, treatment in combination with other drugs to treat cancer. A claim to having invented a MDR modifying drug or mechanisms (here NDGA) is only valid in association with other drugs. Otherwise the molecular and cellular concept would be in conflict with the current best knowledge in oncology research: that is that MDR develops in response to an antineoplastic agent and to overcome such resistance, the antineoplastic agent which created the resistance (or a derivative of the antineoplastic agent) must be administered to overcome it. Along with the agent claimed to overcome MDR resistance (here NDGA). Other mechanisms of MDR exist, however Howell does not elude to them in his application. And even those would require presence of the original antineoplastic agent. An interpretation of Howell that it is intended as a sole therapy would contradict the prevailing theories of treatment of MDR which requires the administration of a sensitizer (in this case NDGA) with the original antineoplastic drug. For a priori existing MDR, the same concepts should apply as for developing MDR under treatment: for a priori MDR, the antineoplastic agent should be given upfront in combination with the MDR modifying agent.

36. In oncology the sequence before-during-after does constitute one and the same regimen for a treatment cycle. As long as the combination of different agents is used within one treatment cycle it is considered that the agents given during that cycle act in concert, hence they are given during one cycle. The timely sequence does not necessarily matter. It is common in oncology to administer agents sequentially within a cycle. The time interval of sequencing also does not necessarily matter as long as it is considered to be one therapy given, e.g. one regimens of a therapy given in one cycle. Howell nowhere described or suggests that NDGA would be given in alternating cycles with antineoplastic agents; in which case a claim for single agent anticancer activity of NDGA might be made. However such interpretation would render Howell useless as a method of addressing MDR.

37. As noted previously, Howell itself rejects single agent antineoplastic activity of NDGA in his application.

38. Huang and al have shown that NDGA and derivatives can have potential single agent activity, that is antineoplastic activity, in solid tumors and hematological malignancies (i.e. leukemias). This is a fundamentally different concept from treatment of the MDR problem by Howell and was in my opinion not anticipated by Howell.

**Rejection of Claims 9-10, 15, 18-20 and 32-33 under 35 U.S.C. § 103 as being obvious over Howell.**

39. I have reviewed the Examiner's Final Office Action of July 1, 2009 in which claims 9-10, 15, 18-20 and 32-33 were rejected as being obvious under 35 U.S.C. § 103 over Howell.

40. The present claims are not obvious over Howell.

41. My statements in paragraphs 30 through 38 are repeated as being directly applicable.

42. In my view Howell requires the administration of a separate neoplastic agent to be operable.

43. On the basis of Howell, one of skill in the art would not expect that leukemia could be treated by the administration of an NDGA derivative alone as described by Howell.

I have read the foregoing Declaration and Office Action Response submitted herewith, and to the best of my knowledge, information and belief, the allegations contained therein are true and correct.

November 30, 2009



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Raoul Tibes

## CURRICULUM VITAE

### PART I: GENERAL INFORMATION

**NAME:** Raoul Tibes

**WORK ADDRESS:** TGen Clinical Research Service  
Scottsdale Clinical Research Institute  
10510 N. 92nd Street, Suite 200  
Scottsdale, AZ 85258  
Phone Office: 480-323-1350  
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### Education

1999	MD, PhD Program Ludwig-Maximilians-University Medical School, Munich, Germany
2000	<i>magna cum laude</i> , PhD ("Doktor der Medizin"), Ludwig-Maximilians-University Medical School, Munich, Germany

### Postgraduate Training

07/00-06/01	Intern in Internal Medicine, New York University Medical Center Residency Program, New York, NY
07/01-06/03	Resident in Internal Medicine, New York University Medical Center Residency Program, New York, NY
07/03-06/06	Fellow in Medical Oncology and Hematology, MD Anderson Cancer Center, Houston, TX
07/04-06/06	Research Fellow, Translational Research in Leukemia, Signal Transduction and Proteomics, Departments of Leukemia and Molecular Therapeutics, U.T. MD Anderson Cancer Center, Houston, TX
01/06-6/06	Fellow in Hematology, MD Anderson Cancer Center and Baylor College of Medicine, Houston, TX, <i>Clinical Rotations in Benign Hematology</i>

### Professional Positions and Academic Appointments

2006-	Assistant Clinical Investigator, Genomics Medicine and Individualized Therapy Center, TGen Clinical Research Service/Scottsdale Clinical Research Institute (SCRI), Scottsdale, AZ
2006-	Associate Investigator, Clinical Translational Research Division and Pharmaceutical Genomics Division, Translational Genomics Research Institute (TGen), Phoenix, AZ
2007-	Director, Hematological Malignancies Program, TGen Clinical Research Service at Scottsdale Healthcare, Scottsdale Clinical Research Institute, Scottsdale, AZ
2007-2008	Member, University of Arizona Cancer Center, Tucson/Phoenix, AZ
2008-	Associate Adjunct Faculty and Member, Mayo Clinic Cancer Center, Scottsdale, AZ

### Licensure



1999-	German Medical License
2000-2003	New York State Board for Medicine, Physician in Training License/Permit
2003-2006	Texas State Board of Medical Examiners, Physician in Training License/Permit
2006-	Arizona Medical License

### **Certification**

2000	USMLE / ECFMG Certification
2003	Board Certified and Diplomate in Internal Medicine, American Board of Internal Medicine
2006	Board Certified and Diplomate in Hematology, American Board of Internal Medicine
2008	Board Certified and Diplomate in Medical Oncology, American Board of Internal Medicine

### **Awards And Honors**

2000	Training Award, European Association for the Study of Diabetes (EASD)
2000	<i>Magna cum laude</i> , Ludwig-Maximilians-University Medical School
2005	Merit Award, American Society of Clinical Oncology (ASCO)
2005	Fellow Award, 58th Annual Symposium on Fundamental Cancer Research, U.T. MD Anderson Cancer Center, Houston, TX
2005	American Society of Hematology (ASH) Merit-Travel Award
2006	ASCO Foundation Merit Award
2006	Young Investigator Award (YIA), ASCO
2008	Personalized Medicine Research Award, IBIS Foundation of Arizona, Phoenix, AZ
2009	2 <sup>nd</sup> Personalized Medicine Research Award, IBIS Foundation of Arizona, Phoenix, AZ

### **Professional Societies**

1999-	Member of the German Association of Physicians
2004-	American Association for Cancer Research (AACR), Member
2004-	American Society of Clinical Oncology (ASCO), Active Junior Member
2008-	American Society of Hematology (ASH), Member

### **Educational Activities - Academic**

2007-	Faculty, Emerging Trends in Oncology, CME-Conferences, Phoenix, AZ
2007-	Faculty for Clinical Rotations for Medical Students of the University of Arizona College of Medicine, Phoenix, AZ
2007-	CME Lectures on AML, Scottsdale Healthcare Hospitals, Scottsdale, AZ
2007-	Mentor for Students accepted in the Helios Scholars Program Internship at TGen, Helios Education Foundation, Phoenix, AZ
2007-	Mentor for Interns volunteering for clinical-translational research projects at TGen Clinical Research Service (TCRS)
2008-	Patient Education and CME Lectures for the Leukemia Lymphoma Society (LLS) Arizona Chapter, Phoenix, AZ
2009-	College of Medicine, University of Arizona, Lectures on Oncology Topics
2009-	College of Medicine, University of Arizona, <i>Mentor for Medical Students</i>
2009-	Wellness Community Arizona, Patient Education Programs.

## **PART II: RESEACH: CLINICAL, LABORATORY, TRANSLATIONAL**

### **CLINICAL TRIAL PROTOCOLS**

## CLINICAL STUDIES

Principal Investigator on 9 Phase I/II studies and Co-Investigator on ~ 30 clinical studies

## PART III: BIBLIOGRAPHY

### Oral Presentations

#### *Invited Lectures*

- 2007 Clinical Science Symposium on "Use of protein arrays to identify therapeutic targets in AML", British Society for Haematology Annual Scientific Meeting, Bournemouth, UK
- 2008 Presidents Guest Lecture "Molecular Abnormalities in Sarcomas – A Paradigm for Targeted Drug Development" at the Annual Meeting of the Musculoskeletal Tumor Society, Phoenix, AZ
- 2008 Genomic Vulnerabilities within the PI3K/Akt Pathway in Acute Myeloid Leukemia Cells, Mayo Clinic, AZ
- 2008 "Controversies in Hematological Malignancies", Symposium on Clinical Challenges in Myelodysplastic Syndromes, Phoenix, AZ
- 2009 "Comparing and Contrasting New Antiangiogenic Tyrosine Kinase Inhibitors", Molecular Oncology: The 6th Vital Sign What Every Oncologist Needs to Know (Conference Director: Daniel Von Hoff), AZ
- 2009 "Identification of Sensitizing Targets to the Hypomethylating Agent 5-Azacytidine in Myeloid Leukemia Cells Using RNAi", Van Andel Research Institute, Grand Rapids, MI
- 2009 "Genomic Characterization of Leukemias: What We Know About Biomarkers and Therapeutic Targets", 6<sup>th</sup> Symposium on Myeloma, Lymphoma and Leukemia, Phoenix, AZ
- 2009 American Osteopathic Association Annual Meeting, AOA Research Conference, "Oncogenomics in Clinical Practice", New Orleans, November 2009

### Oral Presentations

- 2003 "Fanconi Anemia and DNA Repair Mechanisms", Bioimmunotherapy Departmental Conference, U.T. MD Anderson Cancer Center, Houston, TX
- 2003 "EGF-Receptor Inhibition in Non-Small Cell Lung Cancer (NSCLC)", Presented at Dr. W.K. Hong Case Conference – U.T. MD Anderson Cancer Center, Houston, TX
- 2004 "Methylation and Acetylation as Therapeutic Targets in Adenocarcinomas of the Lung", Presented at the Thoracic, Head and Neck Medical Oncology Seminar, U.T. MD Anderson Cancer Center, Houston, TX
- 2004 "Non-Seminomatous Germ Cell Tumors", Presented at the Clinical Genitourinary Case Conference, U.T. MD Anderson Cancer Center, Houston, TX
- 2005 "Use and Utility of Reverse Phase Protein Array (RPPA) for the Analysis of Primary Leukemia Specimens and Hematopoietic Stem Cells", Phase I Clinical Trial Meeting, U.T. MD Anderson Cancer Center, Houston, TX
- 2006 "Intensive Conventional Chemotherapy for High-Risk Aggressive Non Hodgkin's Lymphoma", Citywide Grandrounds, Hematology, Baylor College of Medicine, Houston, TX
- 2007 "Translational Cancer Research – Examples from a Physicians Perspective", Translational Genomics Research Institute (TGEN), Phoenix, AZ

### Publications

1. **Tibes R.**, Trent J., Kurzrock R., Tyrosine Kinase Inhibitors and the Dawn of Molecular Cancer Therapeutics *Annu Rev Pharmacol Toxicol.* 2005;45:357-84.
2. **Tibes R.**, Keating M., Ferrajoli A., Wierda W., Ravandi F., Garcia-Manero G., O'Brien S., Cortes J., Verstovsek S., Browning M., Faderl S. Activity of Alemtuzumab in CD52-Positive Acute Leukemias, *Cancer.* 2006 Jun 15;106(12):2645-51
3. **Tibes R.** et al. Reverse Phase Protein Array (RPPA): Validation of a Novel Proteomic Technology and Utility for Analysis of Primary Leukemia Specimens and Hematopoietic Stem Cells (HSCT), *Mol Cancer Ther.* 2006 Oct;5(10):2512-21.

4. Estey E, de Lima M, **Tibes R.**, Pierce S, Kantarjian H, Champlin R, Giralt S., Prospective feasibility analysis of reduced intensity conditioning regimens (RIC) for hematopoietic stem cell transplantation (HSCT) in elderly patients with acute myeloid leukemia (AML) and high-risk myelodysplastic syndrome (MDS). *Blood*. 2007 Feb 15;109(4):1395-400.
5. **Tibes R.**, Kornblau SM, Qiu Y., Mousses S.P., Robbins C., Moses T., Carpten J.D., PI3K/AKT Pathway Activation in Acute Myeloid Leukaemias is Not Associated With AKT1 Pleckstrin Homology (PH) Domain Mutation, *Br J Haematol*. 2008 Feb;140(3):344-7
6. \*Kornblau SM, \***Tibes R.**, et al. Functional Proteomic Profiling of Acute Myeloid Leukemias (AML) Predicts Response and Survival Outcome. *Blood*. 2009 Jan 1;113(1):154-64 \**Equal contribution*
7. Eisenman KM, Dykema KJ, Matheson SF, Kent NF, DeWard AD, West RA, **Tibes R.**, Furge KA, Alberts AS, "5q- Myelodysplastic Syndromes: Chromosome 5q Genes Direct a Tumor Suppression Network Sensing Actin Dynamics" *Oncogene*. 2009 Jul 13. [Epub ahead of print]
8. D. D. Von Hoff, P. M. LoRusso., C.M. Rudin, J. Reddy, B. Yauch, **R. Tibes**, et al., "Safety and Efficacy of GDC-0449 Hedgehog Pathway Inhibitor in Patients With Locally Advanced or Metastatic Basal Cell Carcinoma in a Phase I Trial", *New England Journal of Medicine*, 2009 Sept 02, [Epub ahead of print]

### Manuscripts in Submission and Preparation

1. **Tibes et al.** "Evidence of Hepatitis C Reactivation under Treatment with Decitabine (*in submission*)
2. **Tibes et al.**, "Patient Willingness to Undergo Pharmacodynamic and Pharmacokinetic Tests in Early Phase Oncology Trials", (submitted to JCO)
3. **Tibes et al.** "RNAi in AML Identifies Sensitizers to 5-Azacytidine that can be Exploited Therapeutically" (*in preparation for Blood*)
4. **Tibes et al.**, "Phase I Study of the Survivin and CDC2/CDK1 Inhibitor Terameprocol in Patients with Advanced Leukemias" (*study completed, data collected, analyzed and manuscript in writing*)
5. **Tibes et al.** "RNAi Synthetic Lethal Kinome Screening with Cytarabine in Myeloid Cells" (*in preparation*)

### Book Chapters

**Tibes R.**, Faderl S., Estey E., Acute Myeloid and Lymphoid Leukemias, Cancer Medicine 7th Edition, Need to Know Oncology – Online Updates 2007, Decker Publishers & American Association for Cancer Research (AACR): [www.aacr.org](http://www.aacr.org)

### Scientific/Laboratory Abstracts

1. **Tibes R.**, Liu Y., Siwak D., Hennessy B., LaPushin R., Mills G.B., Identification of selective inhibition of phospho-S6 ribosomal protein in XK469 sensitive leukemia cell lines using functional proteomic analysis. *Proc Am Asso Cancer Res 2005, Abstract # 3279*
2. Hennessy B.T., Lu Y., Lahad J., Siwak D., **Tibes R.**, Gonzalez-Angulo A, Mills GB., Use of functional proteomics in classification of breast cancer, *Proc Am Asso Cancer Res 2005, Abstract # 3686*
3. Hennessy B.T., Yiling L., **Tibes R.**, Gonzalez-Angulo A., Hortobagyi G.N., Valero V., Mills G.B., Reverse phase protein array in breast cancer classification, *San Antonio Breast Cancer Symposium, 2005*
4. **Tibes R.**, Lu Y., Giles F., Estey E., Hennessy B., Kantarjian H., Kornblau S., Mills G.B. Reverse Phase Protein Array (RPPA): Validation of a Novel Proteomic Technology and Utility for Analysis of Primary Leukemia Specimens, *Presented at the AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics, Philadelphia Nov. 2005*
5. Kornblau S.M., Qiu Y.H., **Tibes R.**, Lu Y., Mills G.B., Time Course Proteomic Profiling Of Signal transduction and Apoptosis Pathways In AML Survivor Cells Using Reverse Phase Protein Lysate Microarray (RPPA) Reveals Differential Effect of Time, Dose and Agent(s), #1219 *Proc Am Soc Hem, Annual Meeting 2005*
6. **Tibes R.**, YiHua Qiu Y., Kevin R.C., Hennessy B., Kantarjian H., Giles F., Estey E., Mills G.B.; Kornblau S.M. Proteomic signatures of acute myeloid leukemia (AML) distinguishes different outcome groups across cytogenetics and identified potential therapy targets. *Poster discussion at the Proc Am Soc Clin Onc 2006. Abstract # 6523*

7. Hennessy B, Gonzalez-Angulo A, Lu Y, **Tibes R**, Sahin A, Hortobagyi GN, Valero V, Mills GB, : Proteomic Prediction in Hormone Receptor-Positive Breast Cancer, *Proc Am Soc Clin Onc 2006. Abstract # 541*
8. **Tibes R.**, Giles F., McQueen T., Bergstrom D.A., Freedman S.J., Andreeff M., Translational in vivo and in vitro studies in patients (pts) with acute myeloid leukemia (AML) chronic myeloid leukemia (CML) and myeloproliferative disease (MPD) treated with MK-0457 (MK), a novel aurora kinase, Flt3, JAK2, and Bcr-ABL inhibitor. *Blood (ASH Annual Meeting Abstracts), Nov 2006; 108: 1362*
9. Kornblau S., Qiu Y., **Tibes R.**, Chen W., Lemker E., Andreeff M., Coombes K.R., Mills G.B., High Throughput Proteomic Analysis of 559 Acute Myelogenous Leukemia (AML) Patient Samples on a Single Slide Using Reverse Phase Proteins Arrays (RPPA): Analysis of Signal Transduction Pathway (STP) and Apoptosis Regulating Proteins, *oral presentation, Blood (ASH Annual Meeting Abstracts), Nov 2006; 108: 107.*
10. Kornblau S., Qiu Y., **Tibes R.**, Lu Y., Mills G.B., Time Course Proteomic Profiling of Signal Transduction and Apoptosis Pathways in AML Survivor Cells Using Reverse Phase Protein Lysate Microarray (RPPA) Reveals Differential Effect of Time, Dose and Agent(s), *Blood (ASH Annual Meeting Abstracts) 2005 106: Abstract 1219*
11. Barrett M.T., Nagel J., Kieffer J.A., Yin H., Que Q., **Tibes R.**, Borad M., Azorsa D.O., Trent J.M, Von Hoff D.D, Mousses M., Clinical vulnerabilities of pancreatic adenocarcinoma genomes, *Proc Am Asso Cancer Res 2007, Abstract # 2932*
12. **Tibes R.**, Meurice N., Anthony S.P., Qiu YH, Kornblau S.M., Mousses M. and Petit J., "Mathematical Models to Identify Combined Protein and Clinical Biomarkers of Response to Induction Chemotherapy in Acute Myeloid Leukemia (AML)", *Blood (ASH Annual Meeting Abstracts), Nov 2007; 110: 2399.*
13. **Tibes R.**, Shanmugam V., Henrichs A., Baker A., Azorsa D., Robeson A., Barrett M, Carpten J., Genomic Vulnerabilities of Acute Myeloid Leukemia Cells, *Poster Presentation, Annual Meeting AACR, #1670, April 2008*
14. **Tibes R**, Choudhary A., Henrichs A., Guled S., Monzon I., Peralta L., Mousses M., and Azorsa D., Identification of Sensitizing Targets to the Hypomethylating Agent 5-Azacytidine in Acute Myeloid Leukemia Cells Using RNAi-Based Functional Genomics Screening, *Blood (ASH Annual Meeting Abstracts), Nov 2008; 112: 2665.*
15. **Tibes R**, Choudhary A., Henrichs A., Guled S., Monzon I., Peralta L., Kiefer, J. and Azorsa D., RNAi-Based Functional Genomics Screening of the Human Kinome Identifies Major Growth Regulating Kinases in Acute Myeloid Leukemia Cells, *Blood (ASH Annual Meeting Abstracts), Nov 2008; 112: 2951.*
16. **Tibes R.**, Bogenberger J. et al, RNAi-based Identification of Novel Sensitizers to 5-Azacytidine in Myeloid Leukemias, *Oral Presentation, European Hematology Association, 14<sup>th</sup> Annual Congress, June 4-7 2009*
17. **Tibes R.**, Bogenberger J. et al., Using RNAi to Identify Novel Molecular Vulnerabilities in Myeloid Leukemias, *Oral Presentation, European Hematology Association, 14<sup>th</sup> Annual Congress, June 4-7 2009*

## Clinical Study Abstracts

1. **Tibes R.**, Delima M., Estey E., Shahjahan M., Champlin R., Non-myeloablative HSCT in elderly AML/MDS patients in 1st CR. *Proc Am Soc Clin Onc 2005. Abstract # 6655*
2. **Tibes R.**, Delima M., Estey E., Pierce S., Champlin R., Giralto S. Prospective Feasibility Analysis of Reduced Intensity Conditioning Regimens (RIC) Hematopoietic Stem Cell Transplantation (HSCT) in Elderly Patients with AML and MDS, #2892, *Proc Am Soc Hem, Annual Meeting 2005*
3. Tolcher A.W., Berk G.I., Fine G.D., Choy G.S., Bearss, D.J., Redkar, S., **Tibes R.**, "MP470, a potent oral Rad51 suppressor is safe and tolerable in first-in-human study", *Poster Presentation, Annual Meeting AACR, #305, April 2008*
4. Wong B. Y., Shapiro G., Gordon M. S., Borad M. J., Eder J. P., **Tibes R.**, Mendelson D. S., Wasserman E., Kawabe T., Sharma S., Phase I studies of CBP501, a novel G2 checkpoint abrogator, alone and combined with cisplatin (CDDP) in advanced solid tumor patients (pts), *Poster Discussion, ASCO Annual Meeting, May 2008*
5. **Tibes R.**, Jimeno A., Von Hoff D.D., Walker R., Pacciarini M.A., Scaburri A., Fiorentini F., Borad M., Jameson G., Hidalgo M., Phase I dose escalation study of the oral multi-CDK inhibitor PHA-848125, *Poster Discussion, ASCO Annual Meeting, May 2008*
6. Anthony S.P., Read W., Rosen P., **Tibes R.**, Park D., Everton D., Tseng B., Whisnant J., Von Hoff D.D., Initial results of a first in man Phase I Study of EPC2407, a novel small molecule microtubule inhibitor anti-cancer agent with tumor vascular endothelial disrupting activity, *Poster Discussion, ASCO Annual Meeting, May 2008*
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